Nephrology

Kidney Function Tests



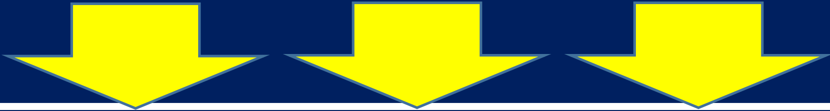
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VISIT…

LANZAROTE Caliente.COM

تحذير هام

هذا الفيديو و جميع الرسوم التوضيحية المتاحة فى هذا الموقع هم تحت حماية قانون الملكية الفكرية



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1. BUN
2. Serum Creatinine
3. BUN/ Creatinine
4. Creatinine Clearance

❶ BUN

Proteins

❶ High protein intake
❷ GIT bleeding
Oesophageal Varices

Tissue Catabolism

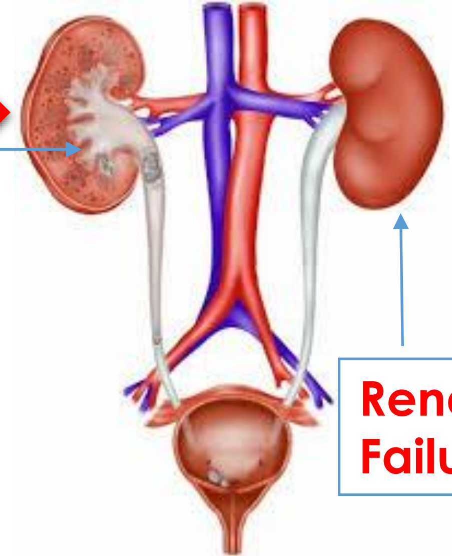
- Fever
- Burns
- Steroids

Ammonia

BUN (Urea)

More time for reabsorption:

1- Decreased Renal Perfusion: Shock- CHF- Dehydration
2- UT Obstruction



Renal Failure

Urine

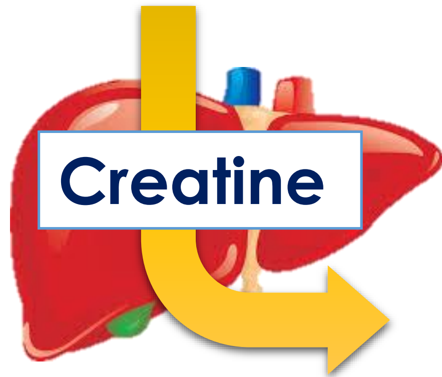
Causes of Decreased BUN

1- Decreased Ptn intake: Malnutrition
2- Increased Dilution of BUN e.g. Increase fluid intake
3- Decrease synthesis by the Liver e.g. LCF
4- Pregnancy

2 Serum Creatinine

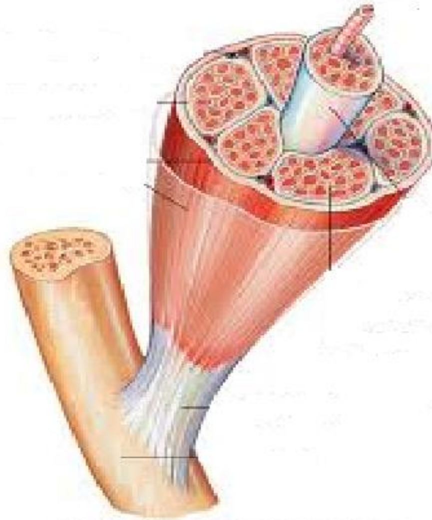
Arginine + Glycine

GAA



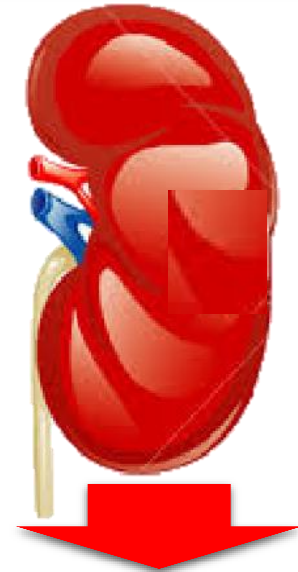
Creatine

Creatinine
(Stored)





Renal Failure



Urine



Rhabdomyolysis

3 BUN/ Creatinine

↑ BUN/ Creatinine

1. High protein intake
2. GIT bleeding: Oesophageal Varices
3. More time for reabsorption:
Decreased Renal Perfusion: Shock-CHF- Dehydration
UT Obstruction
4. Tissue Catabolism
Fever-Burns-Steroids

Renal Failure

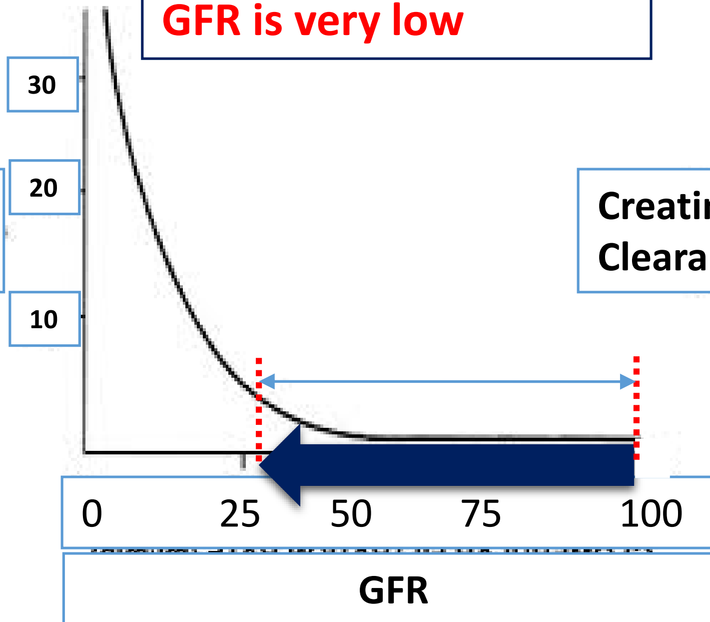
BUN/ Creatinine
10:1

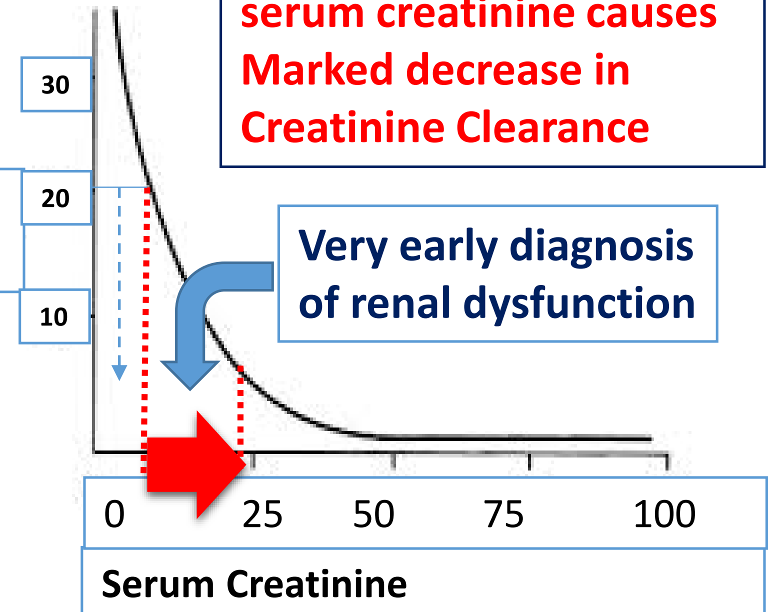
↓ BUN/ Creatinine

Decreased BUN
Increase fluid intake
Low protein diet
LCF
Pregnancy

Increased Creatinine
Rhabdomyolysis
Aspirin
Corticosteroids

4 Creatinine Clearance





Nephrology

Acute Glomerulonephritis



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tarekabdelhamid2000@gmail.com

Definition

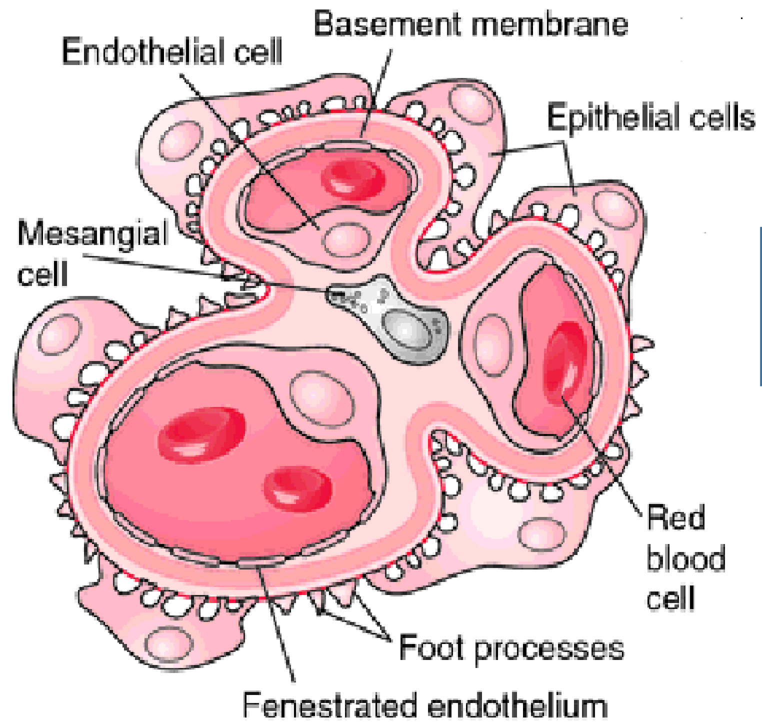
Acute glomerular injury caused most commonly by immune mechanisms e.g.

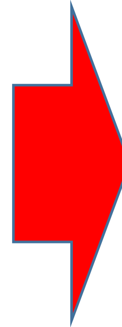
1. Post infections
2. SLE
3. Drugs
4. Malignancy
5. Idiopathic

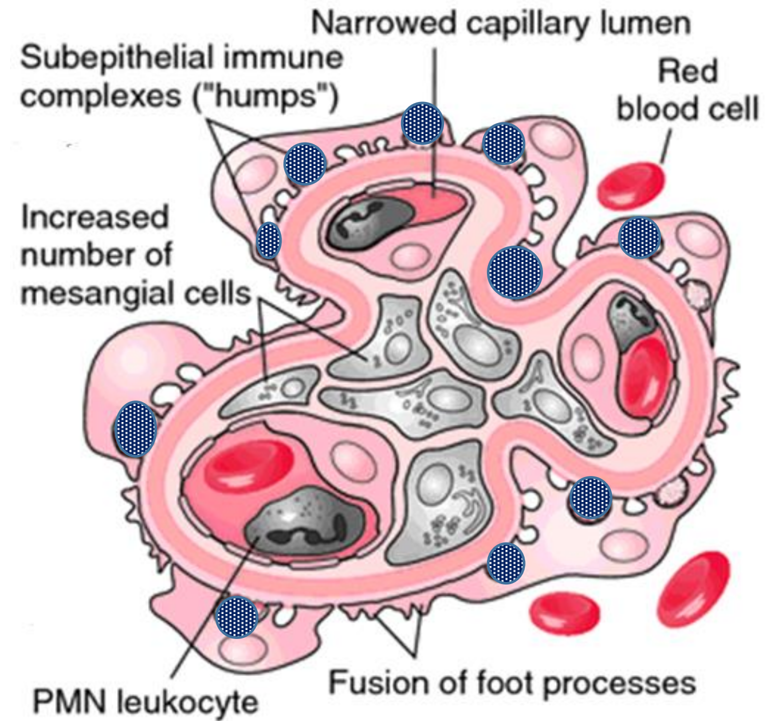
Pathology

Normal

AGN

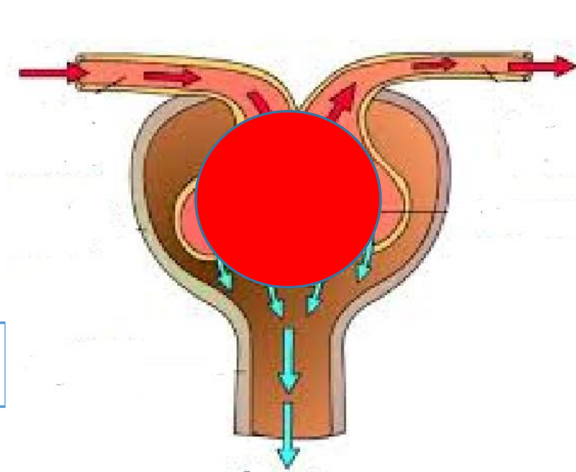






Pathogenesis

Accumulation



H_2O

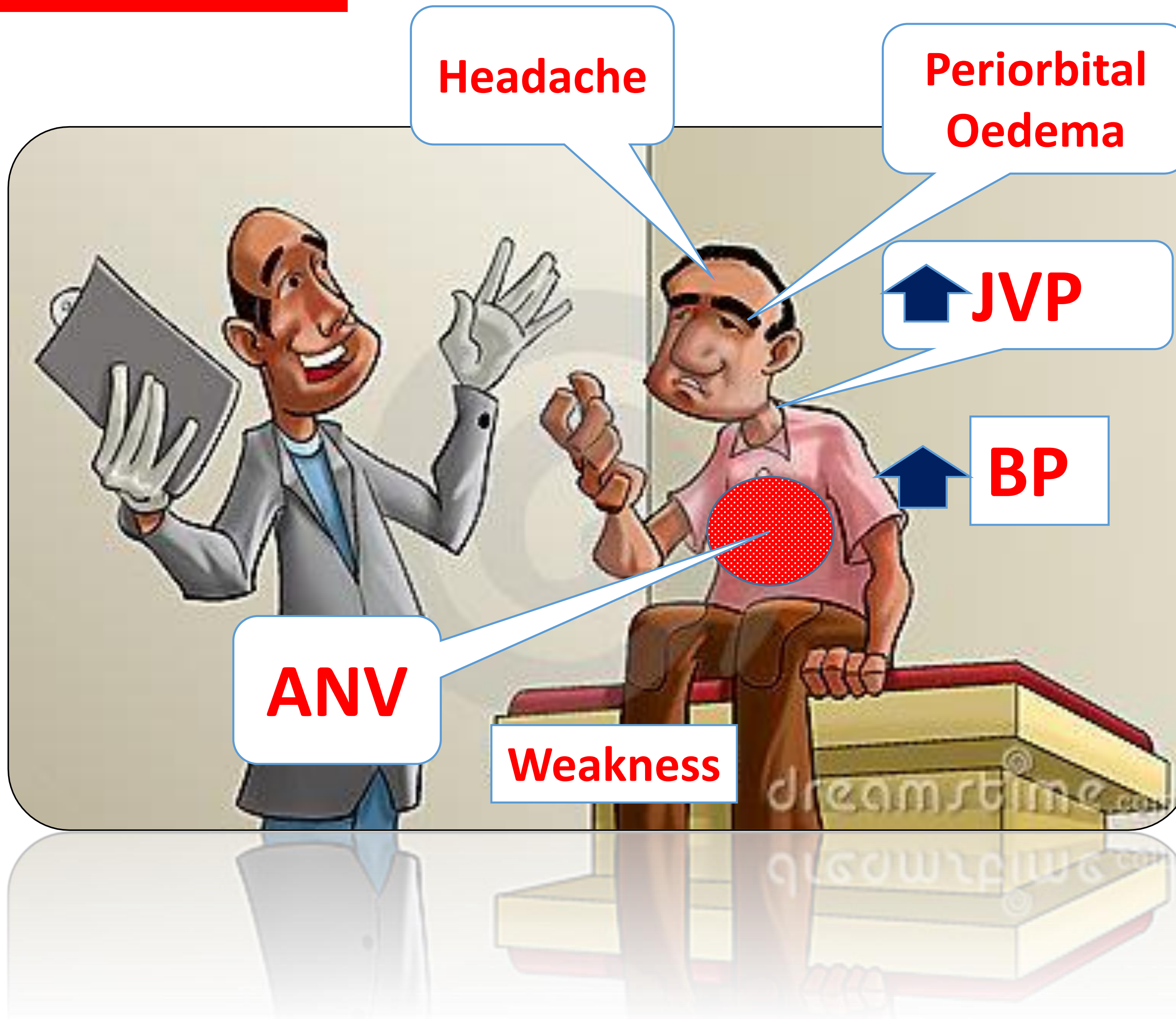
Toxic products

1. ↑ BP
2. Oedema
3. Hypervolemia
   ↑ *JVP*
   Headache

ANV
Muscle weakness

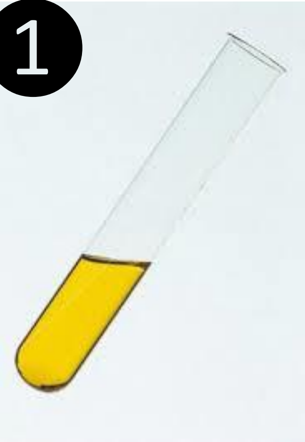
1- Oliguria
2- Microscopic hematuria
3- Red Cell Casts

Clinical manifestations:



Investigations

1



Oliguria
Hematuria
Mild Proteinuria
Smoky Urine

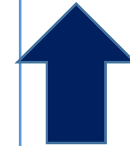
2



Red Cell Casts

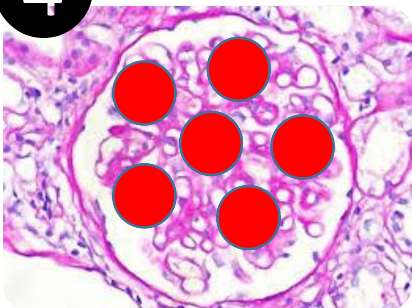
3

Azotemia



BUN and Creatinine

4

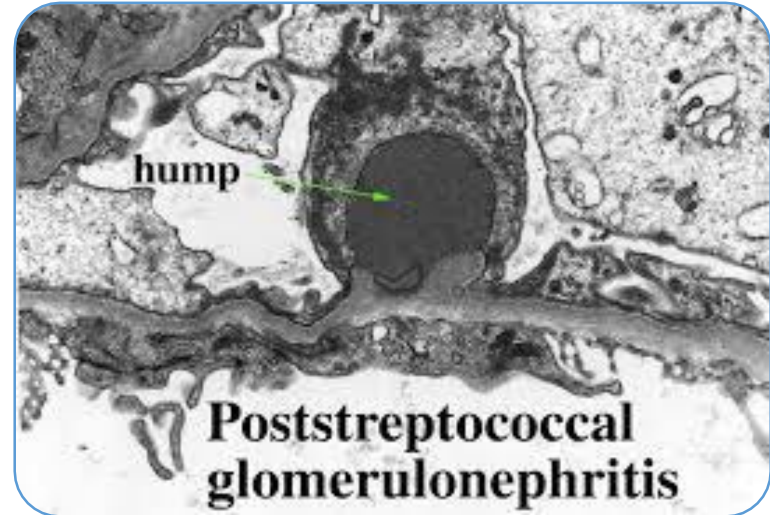


Renal Biopsy:
Immune Complexes

Types

Post Streptococcal GN

❶Caused by Nephritogenic Strain of Streptococci that harbor M Ptn
❷ ++++ESR, ⬆ASOT
❸Low complement level
❹Early use of Antibiotics may be useful
❺Antibodies against the organism (M Ptn) are protective from further attacks.



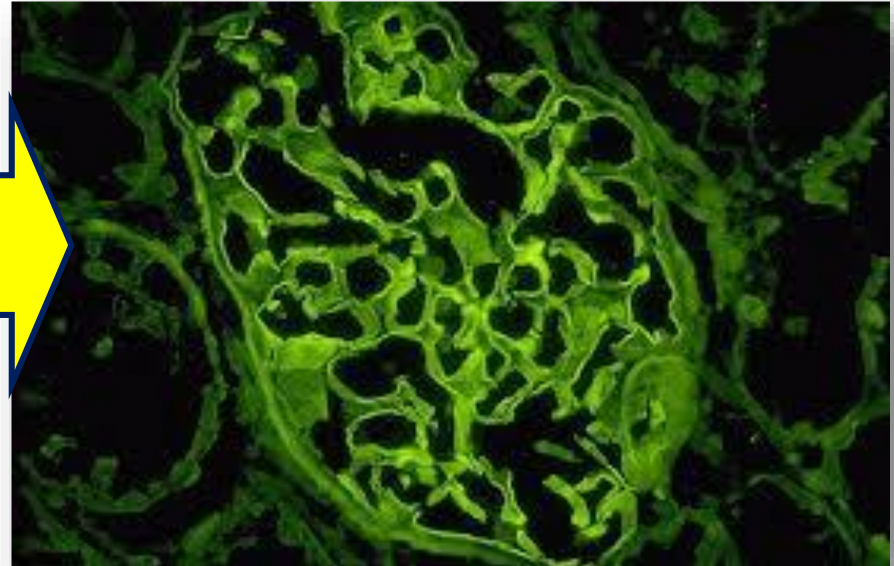
⬆ ESR
⬆ ASOT
⬇ Complement level

Types

Goodpasture syndrome GPS

1- Caused by Anti-GBM Antibodies
2- Associated with Hemoptysis
3- More common in smokers and in Spring
4- Male: Female Ratio 6:1
5- Present mainly as Azotemia
6- Linear Deposits in the glomeruli
6- ttt is by Plasmapharesis

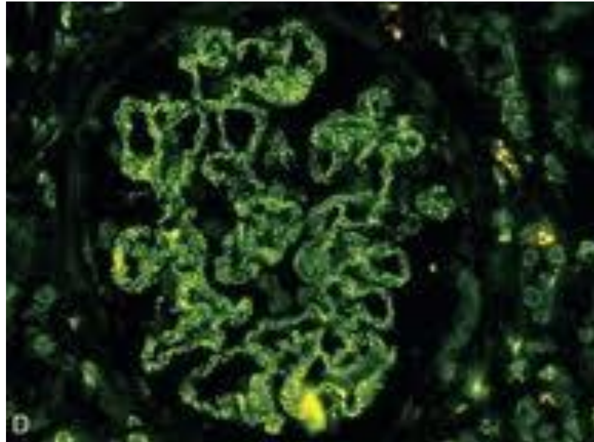
GPS



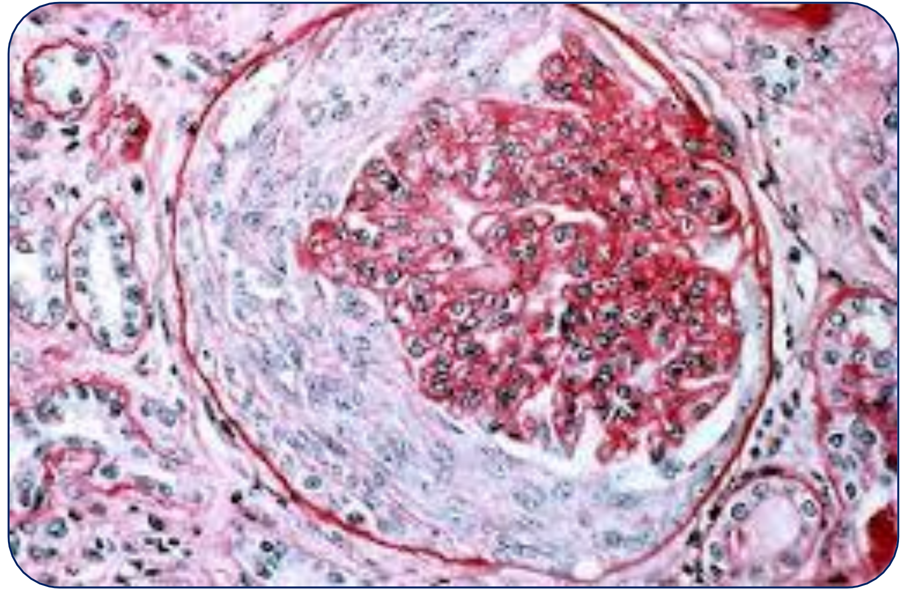
Types

Idiopathic RPGN

1- Characterized by **Circular Deposits** in the glomeruli

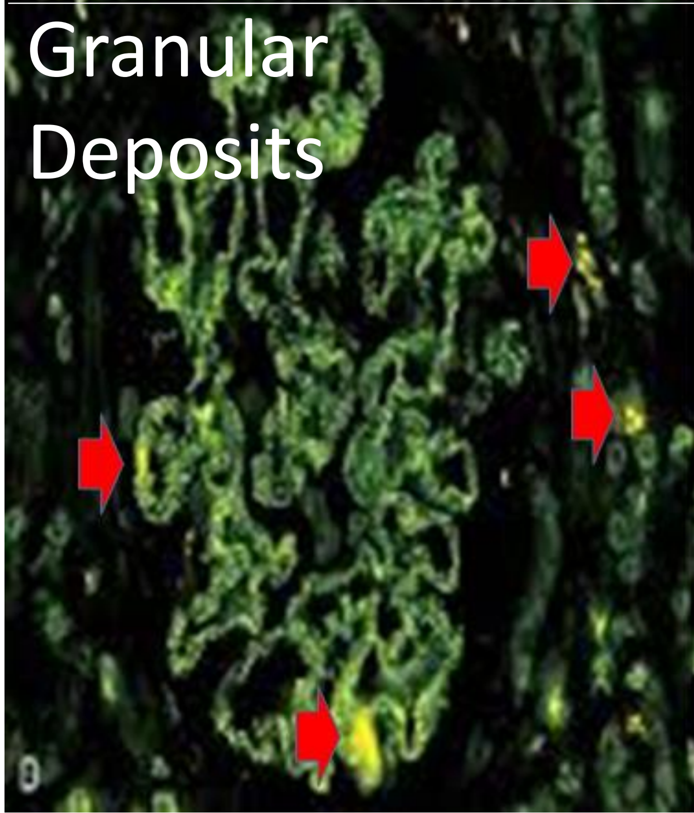


2- **Early ttt by**: Methyl Prednisolone Pulse therapy is Essential

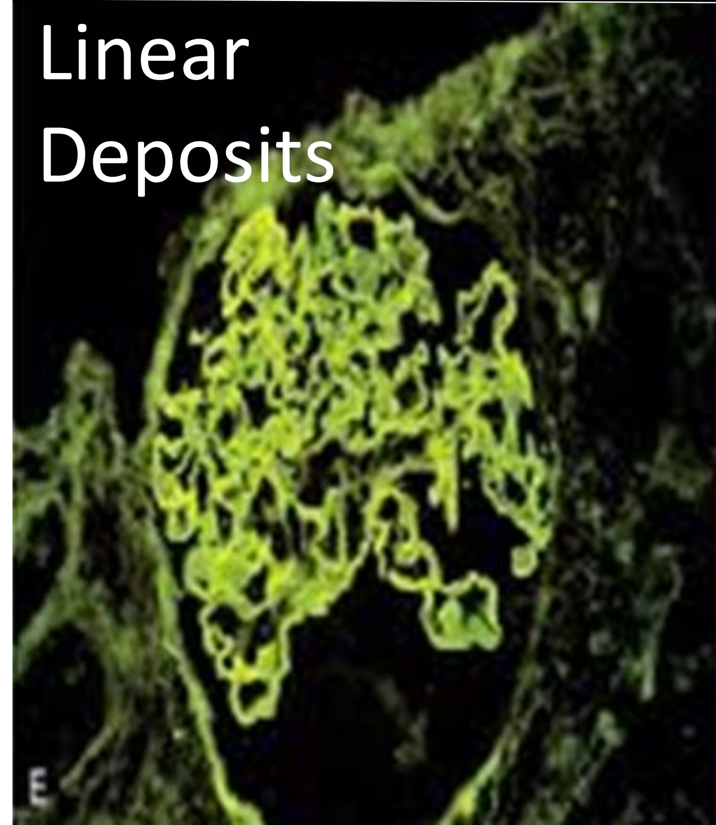


Methyl Prednisolone Pulse therapy

RPGN



GPS



Types

IgA Nephropathy (Burgers Disease)

1- Typically occurs in Young Male
2- Attacks of Recurrent **Hematuria** is the main presenting feature
3- ttt may include Steroids, Fish oil, ACE-I (to decrease the Proteinuria)



Treatment

1- Fluid and Na restriction with Loop Diuretics

2- ⬇ Ptn intake and ⬆ CHO intake

3- Antiplatelet and Anticoagulants

4- STOP….any offending factor e.g. Drugs

5- Specific measures e.g.

Plasmapharesis…(for GPS)

MPPT…(for Idiopathic RPGN)

Steroids + ACE-I + Fish Oil…(for Ig A Nephropathy)

Nephrology

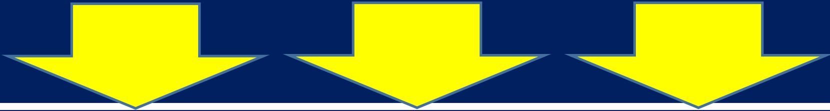
Acute Renal Failure



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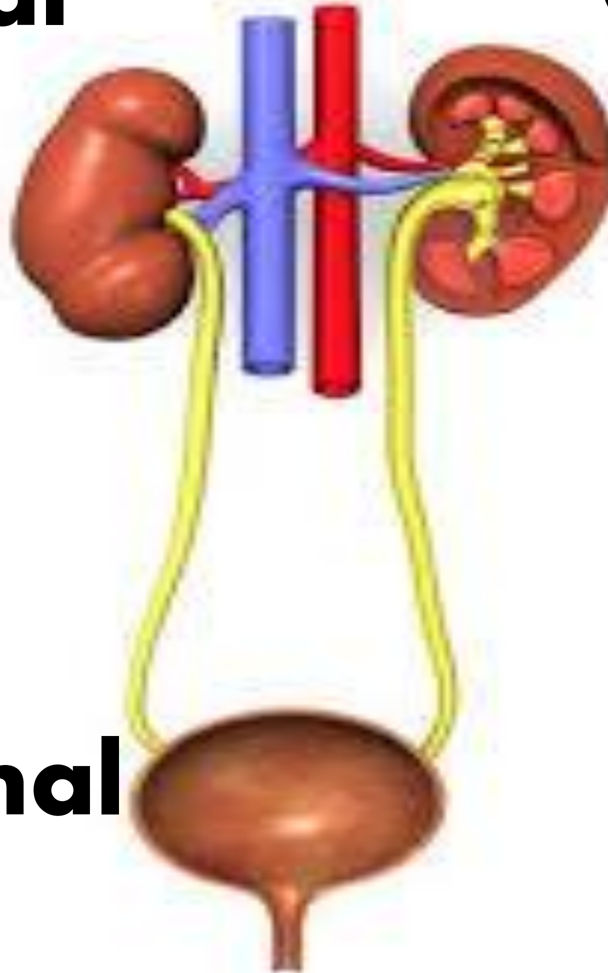
Definition

Rapid decline in kidney functions associated with sudden increase in BUN and Serum Creatinine.

Causes:

❶ Pre-Renal

1- Decreased blood volume: Shock
2-⬇ ECPV: Hypoalbuminemia
3- Excessive VD: Nitrates-Septicemia.
4-Drugs:
Anti-Prostaglandins
ACE-I



❷ Renal

1-++++ GN
2-ATN (Anoxia and Toxins)
3-ATIN (Allergic)
4-Acute deposition of a substance: Uric acid- Oxalate- B J P (Multiple Myeloma)- Sulpha (Children)
5-Rarely; bilateral kidney infection

❸ Post- Renal

<u>Obstruction</u> by stones- Malignancy- BPH
<u>Decreased bladder contractility</u>
e.g. infections - autonomic neuropathy- anticholinergic drugs

Manifestations

↑K



ANV
Weakness
Arrhythmias

Acid Radicals



ANV
Drowsiness
Confusion
Hyperventilation

PO4



↓ Ca — Acidosis → = Ca

Fluids



Features of Hypervolemia

CNS: Drowsiness
GIT: Dyspepsia
Arteries: Hypertension
Veins: Oedema
Na: Dilutional Hyponatremia

Toxic Products

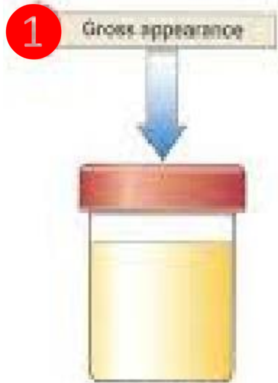


Manifestations of Uremia

ANV- Weakness- Flapping Tremors- Infections (fever?!)- Bleeding tendency

Investigations

1 Urine



Oliguria less than 400 CC/day in many cases

Plus

Casts

1- Brown granular casts……ATN
2- Empty casts………Pre-renal causes
3- Eosinophilic casts…..ATIN
4- Red Cell Casts…..AGN

2 Blood

Rapid rise of Serum Creatinine More than 1mg/dl/day

- ⬆ K
- ⬆ Metabolic Acidosis
- ⬆ BUN
- ⬆ PO4

3 Ultrasonography



To exclude Post-Renal causes



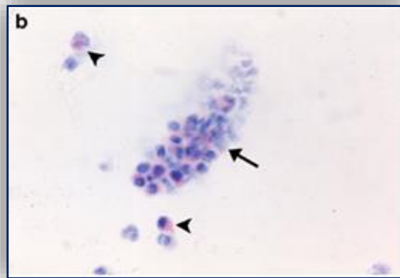
Brown granular casts

Acute Tubular Necrosis



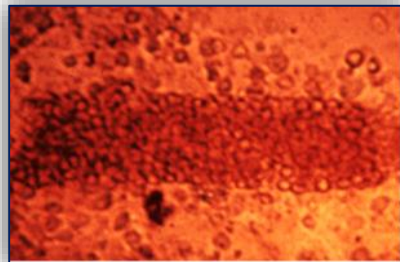
Empty casts

Pre-renal causes



Eosinophilic casts

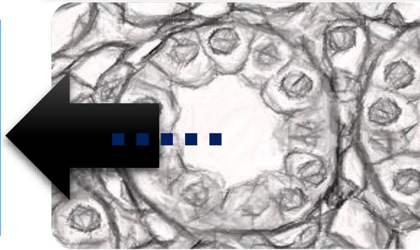
ATIN

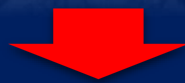
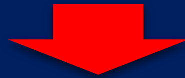


Red Cell Casts

AGN

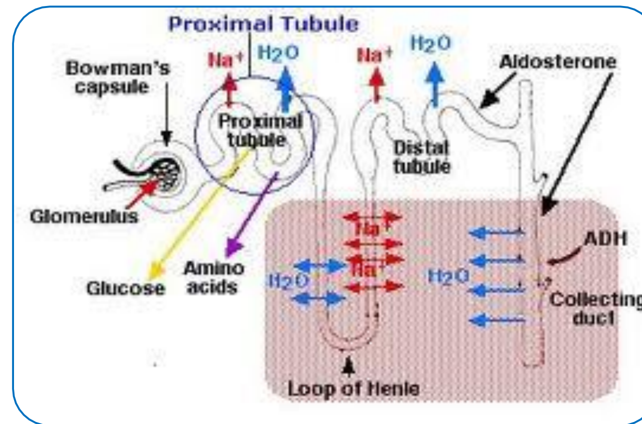
1-Na
2-H20
3-Urea



	Pre-Renal	Renal
❶ FENa		
❷ Urinary Sodium		
❸ U/P Osmolality		
❹ U/P Creatinine		
❺ Oedema	Easier	
❻ Improvement	Rapid Improvement	

Note

ATIN



❶ Typically occurs as an **Allergic** Reaction to drugs e.g. Penicillin -NSAI and -Thiazide & Loop diuretics .

❷ **Fever-Rash-Eosinophilia**

❸ **Acidosis** and electrolyte disturbance <u>disproportionate</u> to the decreased GFR.

Treatment (1)

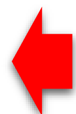
❶ Relief of the cause

❷ Correction of the Metabolic Derangements

❸ Dialysis (e.g. Symptoms of Uremia)

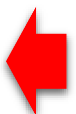
Treatment (2)

↑K



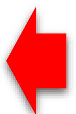
Glucose + Insulin
HCO3
IV $CaCl_2$
No K intake-K exchange resin-Sorbitol

Acid Radicals



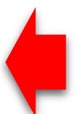
Na HCO3……ONLY if
1. HCO3 less than 10 mEq/L
2. PH less than 7.2
3. ++++K

PO4



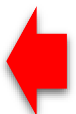
Al (OH)3 GEL TO decrease PO4 absorption.

Fluids



Fluid Restriction according to Urine Volume and daily weight.

Toxic Products



Dialysis
Glucose *plus* Essential amino acids
DDAVP to # Hematemesis

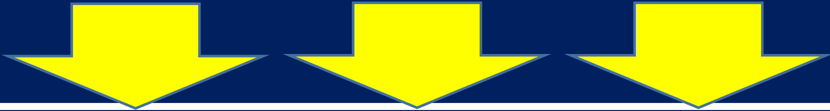
Nephrology

Chronic Renal Failure



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tarekabdelhamid2000@gmail.com Phone: + 1-804-829-8611 (USA)

Definition

Gradual and Progressive decline in GFR secondary to irreversible nephron loss.

Aetiology

A Analgesic Nephropathy- APKD- Amyloidosis

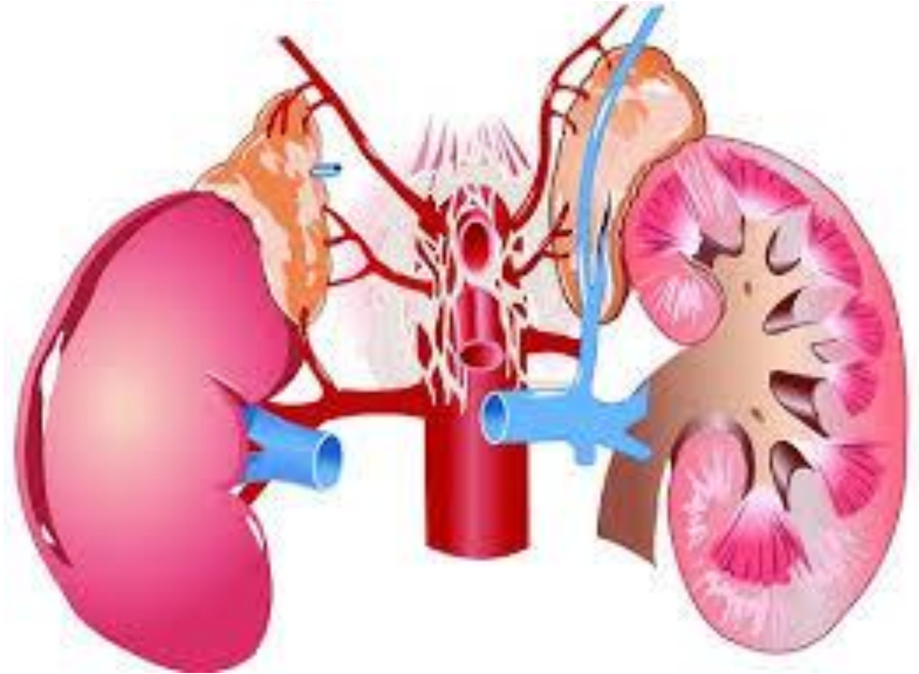
B ⬆ BP BPH Plumbism

C CGN CPN CTIN

D DM

E *Extra*

VUR-Stones-RVT



Manifestations

❶ Accumulation of toxic metabolites



ANV- Hypogonadism- Peripheral Neuropathy- Infections- Acidosis- K- Uric acid- Nail changes

❷ Erythropoietin production



Anemia

❸ Renin Production



↑ Blood Pressure

❹ Accumulation of PO4





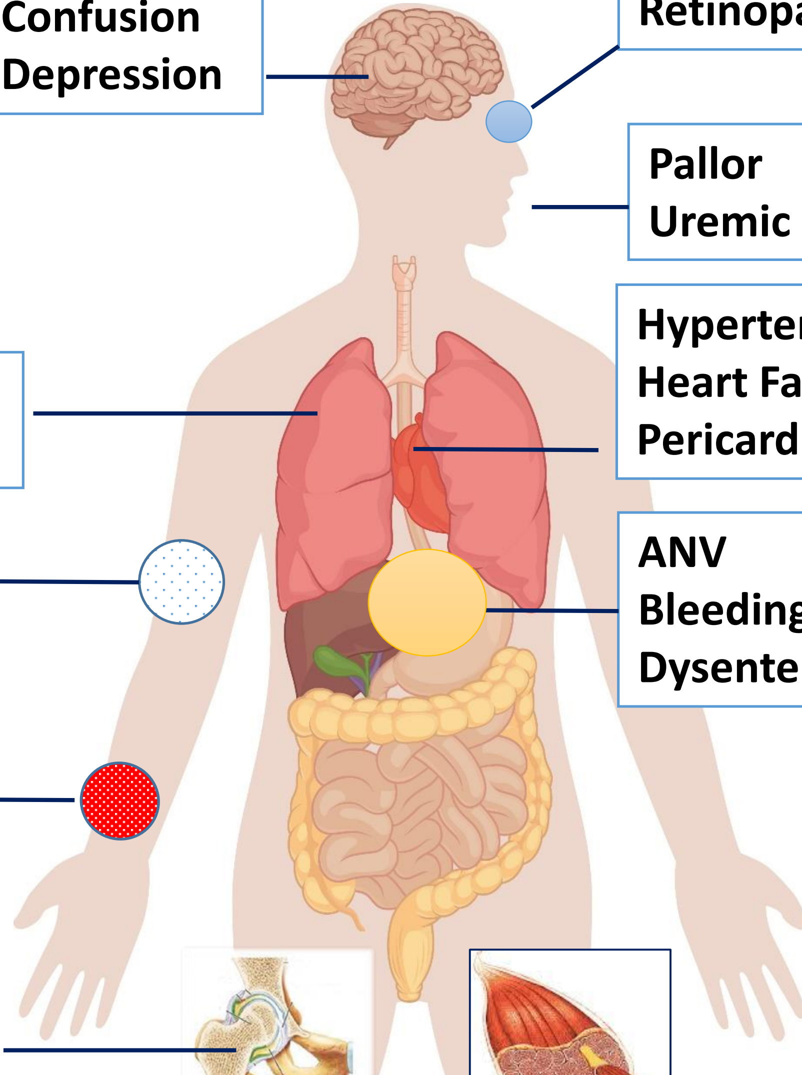
Lack of Vitamin D activation

❺ Inability to concentrate urine



Fixed Specific Gravity (Isothenuria)

Manifestations



Weakness
Headache
Confusion
Depression

Band Keratopathy
Hypertensive Retinopathy

Pallor
Uremic odor

Acidotic breathing
Hemoptysis

Hypertension
Heart Failure
Pericarditis

+++ Itching

ANV
Bleeding
Dysentery

Ecchymosis

Osteomalacia
Fractures
OFC

Cramps



Band Keratopathy



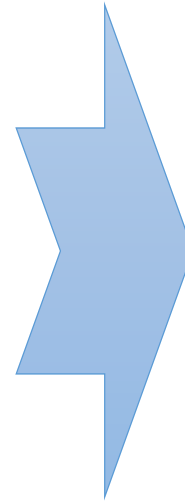
Nail changes



Bone damage



Anemia



Chronic

Investigations

❶ Urine



Fixed Specific Gravity with Broad Casts





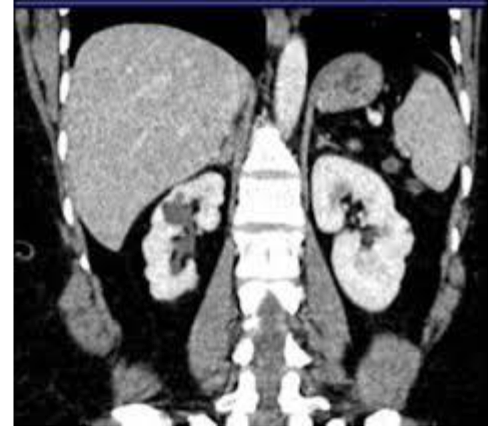
❷ Blood

Progressive and slow elevation of Serum BUN & Creatinine

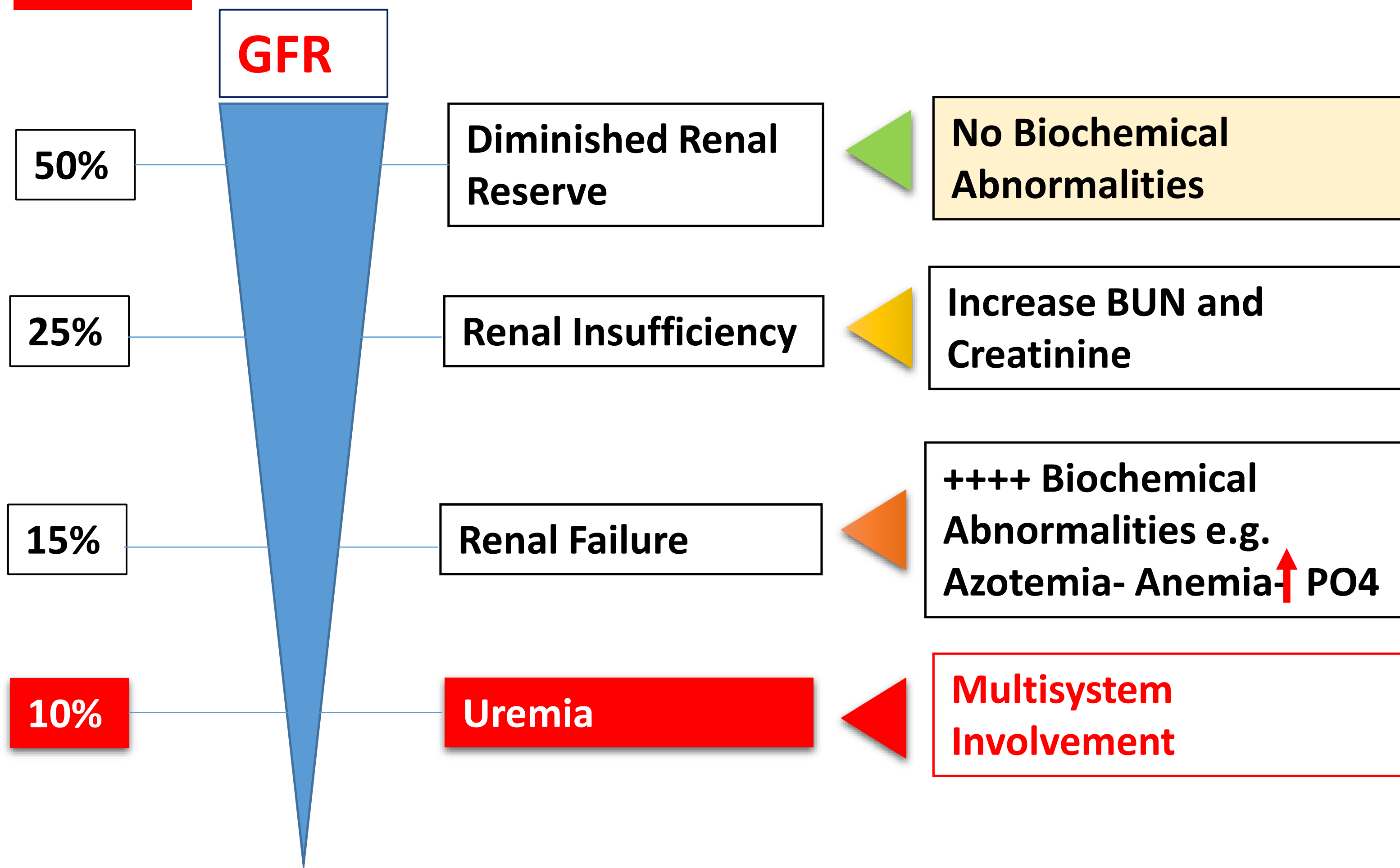
- ↑ K
- ↑ Metabolic Acidosis
- ↑ Uric Acid (?!)

Anemia

❸ Ultrasonography

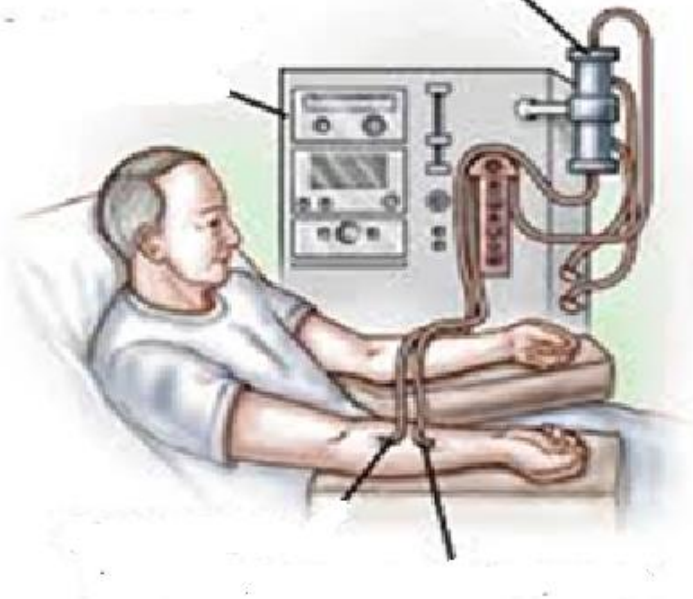


Bilateral Shrunken Kidneys


Stages
GFR
50%
Diminished Renal Reserve
No Biochemical Abnormalities
25%
Renal Insufficiency
Increase BUN and Creatinine
15%
Renal Failure
++++ Biochemical Abnormalities e.g. Azotemia- Anemia- ↑ PO4
10%
Uremia
Multisystem Involvement

Dialysis is the main line of therapy

Note: If you find functional fluid, acid-base, and electrolyte disturbances at a moderately reduced GFR you MUST consider CTIN (?! **Reversibility**)



Nephrology

Nephrotic Syndrome



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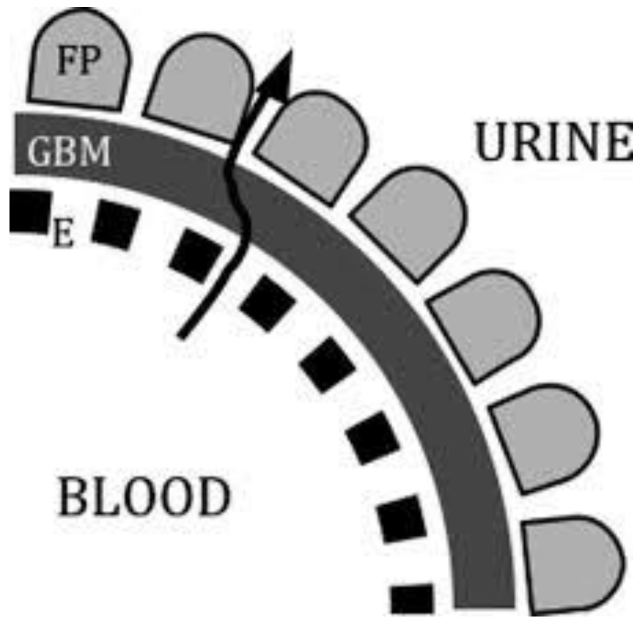
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tarekabdelhamid2000@gmail.com

Definition

A syndrome characterized by heavy proteinuria, Hypoalbuminemia, and Oedema 2ry to damage of the GBM (Most commonly due to glomerulopathy)



Types:

1. Minimal change Lesion
2. Focal glomerular sclerosis
3. Membranous Nephropathy
4. Membranoproliferative GN

Pathogenesis

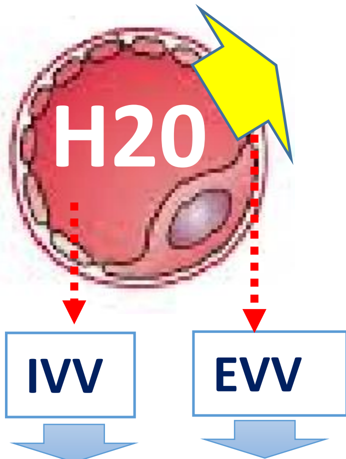
Defect in GBM

Loss of proteins in Urine

Albumin

Globulins

Other proteins



IVV

EVV

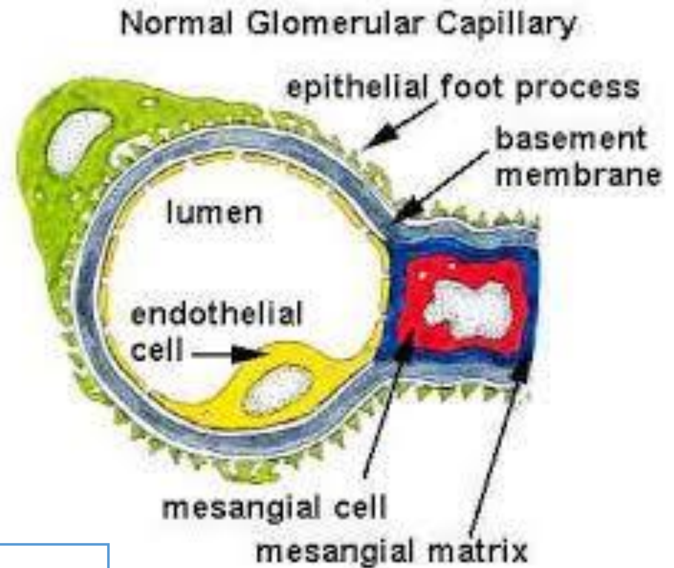
Several Manifestations depending on the tissue.

Infections

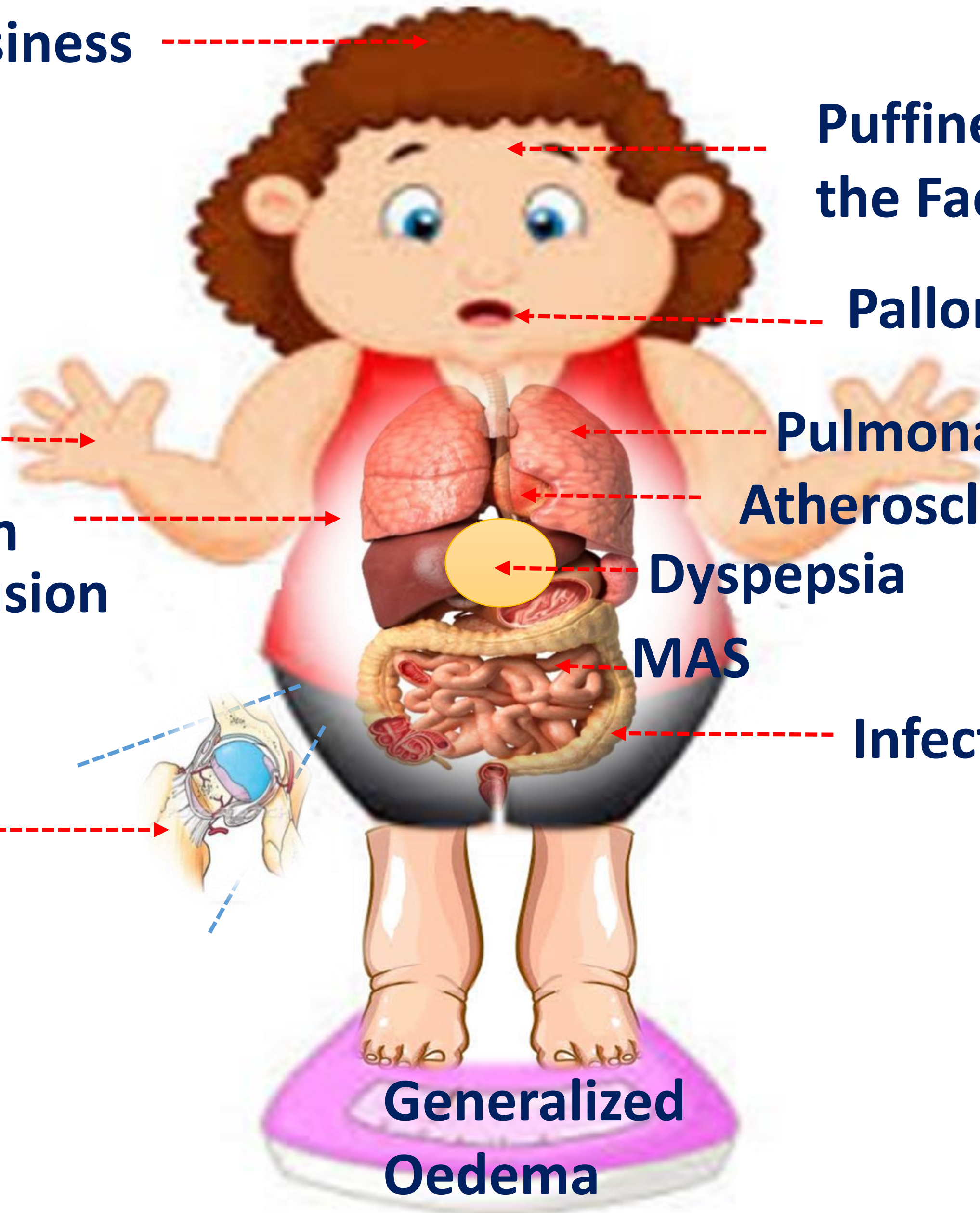
1- AT3…..Coagulopathy

2- Vitamin D-binding globulins……..Bone Damage

3-Transferrin….IDA



Manifestations



Investigations

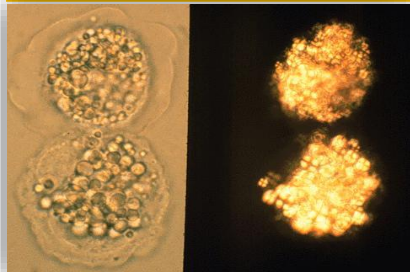
1 Urine



Proteinuria
More than 3.5 g/1.73 meter square per 24 hours.

Plus

Fatty Cast



2 Blood

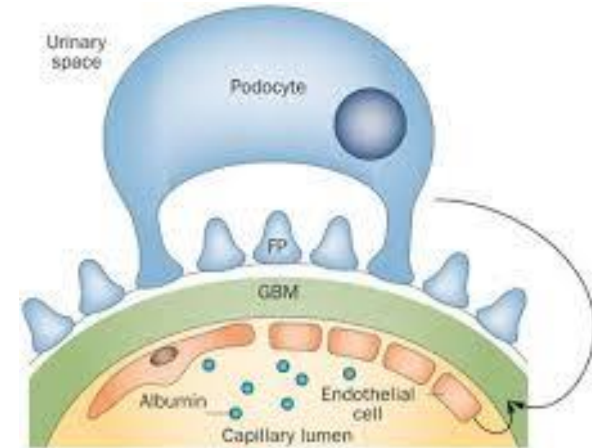


Albumin



Cholesterol

4 Renal Biopsy



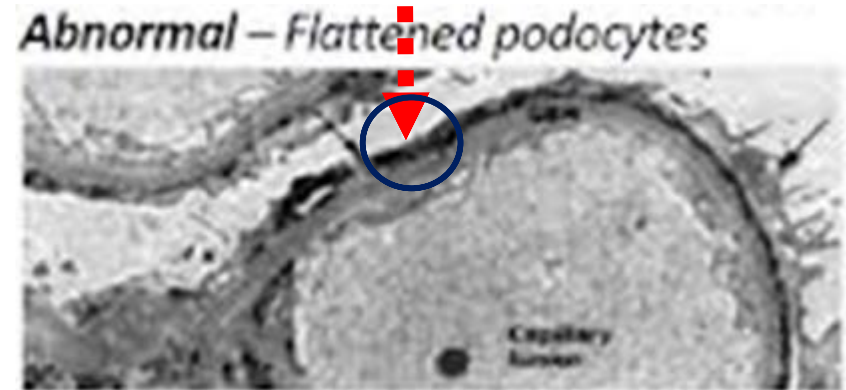
To detect the cause

Types

❶ Minimal change Lesion

1- Typically occurs in **Children**
2- **Fusion of Foot Processes** on EM
3- Usually NO Renal Failure
4- Dramatic response to Steroids (plus Cyclophosphamide)
5- Can be associated with: Allergy- NSAI- Hodgkin Disease (in adults)





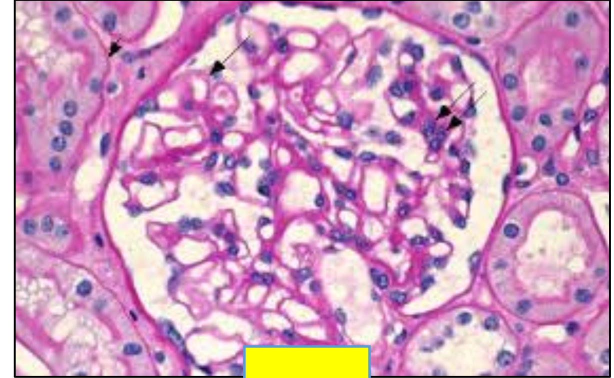
Types

❷ Focal glomerular sclerosis

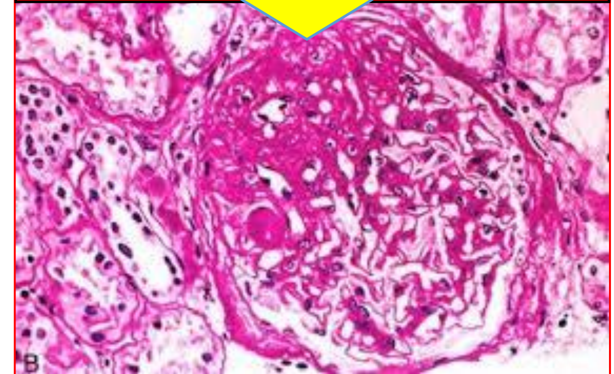
CRF

Associated with Heroin abuse-HIV infections- and VUR

Normal glomerulus



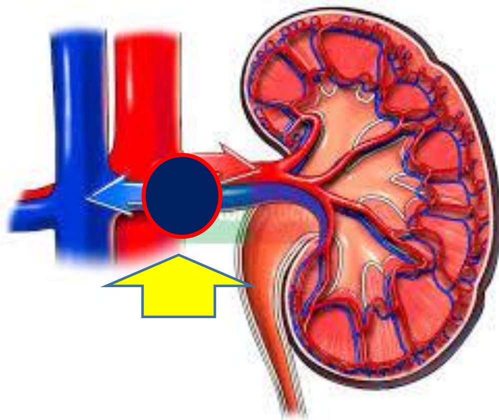
FGS



Types

❸ Membranous Nephropathy

1- Increased incidence of RVT



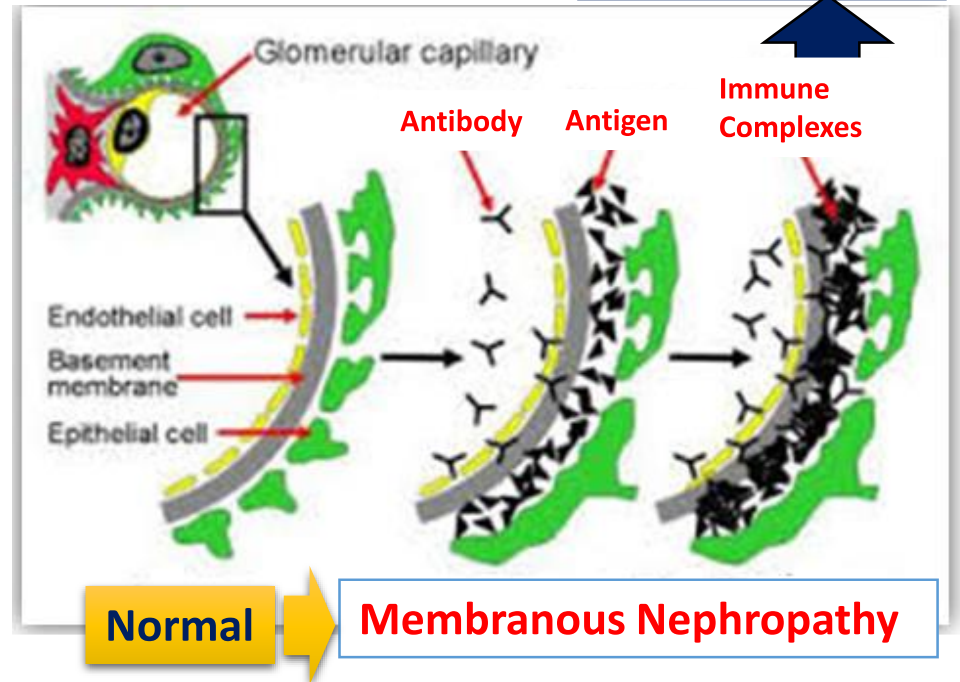
2- Subepithelial deposits on EM

3- Associated with:

Lung Cancer

D-Penicillinase

SLE



Types

❹ Membranoproliferative GN

Type I

Subendothelial deposits by EM

Type II



C3NEF

Associated with:

❶ C3NEF

(an IgG with Properdine like activity ……

Alternative compliment pathway

❷ Dense deposits inside the GBM by EM

Treatment

1- Ensure enough **Protein** intake 1g/kg/day

2- ↓ **Na** intake to diminish the Oedema

3- Use **Diuretics** to treat the Oedema (but notice that they may precipitate pre-renal azotemia especially in patients with Low IVV)

4- ttt of the cause if possible e.g. Steroids for Minimal Change Lesion